

## Another Piece of the Obesity–Environment Puzzle

### Potential Link between Inflammation and POP-Associated Metabolic Diseases

Epidemiologic research suggests that persistent organic pollutants (POPs) in the environment may contribute to the development of obesity and features of metabolic diseases, such as elevated triglyceride levels, glucose intolerance, and cardiovascular disease. POPs include dioxins and furans—with 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) being the most potent of these—and polychlorinated biphenyls (PCBs). Due to their lipophilic nature, these compounds accumulate in adipose (fat) tissue, the site of many endocrine and metabolic functions that are disrupted in metabolic diseases and obesity. A new study outlines a mechanism by which inflammation could play a central role in POP-associated metabolic disease [*EHP* 120(4):508–514; Kim et al.].

TCDD influences genes underlying various cellular processes by binding the aryl hydrocarbon receptor (AhR) and has been shown to stimulate production of enzymes involved in the metabolism of environmental agents. Other halogenated dioxins and furans, as well as coplanar PCBs, may trigger similar effects.

Inflammation, one of the processes affected by these chemicals, is a significant factor in many diseases. Chronic low-grade inflammation

in adipose tissue alters its biological function, potentially leading to insulin resistance and other health problems.

In this study, researchers treated both immature precursor cells and mature adipocytes (fat cells) that developed from them with TCDD, dioxin-like PCB126, and non-dioxin-like PCB153. For the *in vivo* portion of the study, wild-type and AhR knockout mice were injected with a single dose of TCDD or corn oil (control).

Mouse adipose tissue was examined after treatment to determine the size of the adipocytes and the presence of macrophages (an indication of inflammation). Gene expression analysis determined the numbers and types of up- and downregulated genes in both cultured cells and adipose tissue, with those involved in inflammation being the most significantly upregulated. However, the PCB153-associated gene regulation pattern was unlike the patterns for PCB126 and TCDD, whose effects could be blocked by an AhR antagonist *in vitro*.

The inflammatory response was strongly induced in precursor cells and to a lesser degree in adipocytes, and this response was mediated through the AhR. The findings were confirmed in the rodent tissue and are congruent with epidemiologic studies. Caution is required in extending the findings to human health, however, because the rodent exposure regimen was dissimilar from typical human exposures. Nevertheless, this study highlights what may prove to be one of the major mechanisms by which POPs affect disease development.

**Julia R. Barrett**, MS, ELS, a Madison, WI–based science writer and editor, has written for *EHP* since 1996. She is a member of the National Association of Science Writers and the Board of Editors in the Life Sciences.

## Optimizing Insecticide Resources

### Global Trends in Vector Control

Although insecticides have helped lower worldwide rates of infectious disease, the use of these chemicals must be monitored and controlled to avoid pest resistance and minimize associated risks to human health and the environment. Researchers have conducted a comprehensive assessment of trends in the global use of insecticides for vector-borne disease control over the last decade and found that the potential for overuse of pyrethroids could result in a loss of the effectiveness of long-lasting insecticidal nets (LNs), which have been one of the most successful tools for controlling malaria to date [*EHP* 120(4):577–582; van den Berg et al.].



A health officer in Kolkata, India, fumigates to prevent the spread of mosquito-borne diseases in 2005.

The research team analyzed data from 125 countries that used organochlorines, organophosphates, carbamates, and pyrethroids for vector control between 2000 and 2009. Application methods were classified as residual spraying (spraying interior and peripheral surfaces of houses), space spraying (spraying exterior spaces), treatment of nets (not including factory manufacturing of LNs, which contain pyrethroids), and larviciding (treating aquatic breeding sites of mosquitoes with insecticides). The team conducted 2 analyses of insecticide use for each country, covering a 10-year and an annual average.

The main diseases targeted by insecticide use were malaria, dengue, leishmaniasis, and Chagas disease. The organochlorine DDT was by far the most used insecticide in terms of quantity (71%), with India accounting for most applications (82%) and African countries accounting for the rest. Globally the use of DDT has not changed substantially since the Stockholm Convention on Persistent Organic Pollutants was established in 2004, although use increased sharply in Africa until 2008.

Pyrethroid use in residual and space spraying accounted for the greatest surface or area covered (81%), with most use occurring in the Americas, although an increase occurred in African countries. Global pyrethroid use has increased steadily since 2004, mainly due to increased use for residual spraying, a factor the authors warn could promote resistance among mosquitoes, negatively affecting the long-term effectiveness of treated bed nets. They warn that “in areas where resistance genes have already spread, immediate implementation of resistance management is required to preserve the effectiveness of available tools, including LNs.”

The main limitation of the study is countries’ potential lack of capacity for reporting insecticide use, especially regarding dengue control (which tends to be less structured than malaria control). Even so, the data collected can help guide global strategies on vector control and resistance management.

**Tanya Tillett**, MA, of Durham, NC, is a staff writer/editor for *EHP*. She has been on the *EHP* staff since 2000 and has represented the journal at national and international conferences.

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